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Heat-related deaths in Adelaide, South Australia: Review of the literature and case findings – An Australian perspective

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ABSTRACT

Heat waves are not uncommon in Australia, but the event of 2009 was particularly severe and ranks third of the 21 recorded heat wave events in south-eastern Australia in terms of the resulting mortality and morbidity. This is a review of Coronial autopsy findings in South Australia (which has an area of nearly 1 million square kilometres with a population of 1.6 million that predominantly resides within the region of the capital: Adelaide) during the period of the 2009 heat wave. Fifty-four post-mortem examinations were performed on cases in which exposure to high ambient temperature was regarded as having caused or significantly contributed to the death. The findings (including results of toxicological and biochemical analyses, where available) are reviewed and compared with the post-mortem examination findings in 22 deaths over the same period not attributed to the effects of heat. There were no specific autopsy findings that distinguished heat-related from non heat-related deaths. The lack of specific post-mortem findings increases the reliance on scene investigation in order to be able to categorise a death as being heat-related. A checklist for scene investigators is proposed in order to assist with collection of relevant data to assist the Coronial investigation process.

1. Introduction

The Australian climate can be harsh and multiple deaths have been associated with heat waves in Australia throughout the years. A heat wave can be defined as a period of at least three days during which the combined effects of excess heat and heat stress is unusual with respect to the local climate.[1] Of 21 recorded heat wave events in south-eastern Australia since 1985, the heat wave of 2009 resulted in an estimated 404 deaths, which ranks it third in severity being surpassed only by events in 1895/6 and 1938/9.[2]

The human body has a thermoregulatory set point (which is around 37 °C, depending on site of measurement[3]). Ability to dissipate heat can be influenced by factors including age, medical conditions, environment, clothing, mental state and medications. Compensatory mechanisms to reduce body temperature include vasodilatation, which increases the flow of blood to the skin thereby increasing radiant heat loss and sweating leading to increased heat loss by evaporation.[4] Of the four ways in which heat can be dissipated from the body (conduction, convection, radiation and evaporation), evaporation from sweating is the body's most effective method of cooling.[5] Heat stress can be defined as the perception of discomfort due to exposure to a hot environment.[4] The condition of heat oedema refers to swelling of dependent regions due to vasodilation and orthostatic pressure in unacclimatised individuals. Heat syncope can occur as a result of orthostatic hypotension. Heat exhaustion results from exposure to heat causing an elevation of body core temperature above 38 °C, but below 40.5 °C with volume depletion due to sweating without adequate fluid replacement. Heat stroke is a serious condition in

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which there is a body core temperature above 40.5 °C with nervous system dysfunction resulting in confusion and impaired conscious level[6]; it can be divided into classical and exertional forms.[5] Heat stroke can result in multi-organ dysfunction with derangements including rhabdomyolysis, acute renal failure, coagulopathy, pulmonary oedema, myocardial injury, cardiac arrhythmias, and brain dysfunction including seizures and/or encephalopathy.[4,5] These derangements alone or in conjunction with pre-existing disease can cause death.

The National Association of Medical Examiners (NAME) has issued a statement listing criteria for heat-related deaths[7]; their recommended definition of heat-related death is, "a death in which exposure to high ambient temperature either caused the death or significantly contributed to it." This is an acknowledgement of the difficulty in fulfilling the criteria required for the clinical diagnosis of heat stroke[8,9] (core temperature above 40.5 °C or higher with mental confusion) as records of core temperature measurement and cognitive function are usually not available unless death has followed admission to a medical facility. In the absence of such data, the NAME committee would recommend diagnosing heat-related death based on a history of exposure to high ambient temperature when other causes have been excluded. This may be achieved by a review of the circumstances around the death with records of the ambient temperature. Others[10] have also used the concept of designating a death as heat-related based on occurrence during a heat wave when other causes are excluded. It has also been proposed that using the Excess Heat Factor (EHF), which is derived from meteorological data can provide evidence of a period of significant heat stress expected to be associated with heat-related morbidity and mortality.[11]

This review sought to review the findings in cases subjected to post-mortem examination at direction of the Coroner during a heat wave in South Australia that extended from 26th January to 7th February 2009 and the criteria used in arriving at a diagnosis of heat-related death.

2. Methods

Deaths that occurred during the period 26th January to 7th February 2009 that had been reported to the Office of the State Coroner for South Australia were reviewed. Deaths regarded as heat-related were identified with the assistance of the State Coroner's Office. Natural deaths not regarded as heat-related during the period were also collected. The circumstances, scene investigation reports and post-mortem findings were reviewed for the heat-related deaths.

3. Results

During the period 26th January to 7th February 2009 a total of 58 deaths were reported to the Coroner in which exposure to high ambient temperature was regarded as having caused or significantly contributed to the death Fifty-four had a post-mortem examination; death could be attributed to the effects of heat alone in 16 cases. In the remaining 38 cases, heat was regarded as factor in causing death from pre-existing natural disease, which comprised 32 cases with cardiac disease (ischaemic heart disease, cardiomegaly, myocardial fibrosis and/or hypertensive heart disease); 2 with combined cardiac and pulmonary disease (ischaemic heart disease and emphysema); 3 with pulmonary disease (1 pneumonia, 1 lung carcinoma, 1 emphysema) and 1 deceased with anorexia. Of the 42 deaths that occurred indoors, 15 had been found dead in bed. Eleven had air conditioners, but only 5 were recorded as being on. The presence or absence of a fan was noted in 31 cases, with 21 having a fan (but it was not recorded if the fan was operating). The interior temperature was recorded as 30–40 °C, hot or very hot in 22 cases and stifling, extremely hot or greater than 40 °C in 8 cases. It was recorded that 9 of the deceased had lived alone and 3 had at least one other person present, but this data had not been captured for 30 of the deaths that had occurred indoors. There was no record of what activities deceased had been engaged in around the time of their death. For 43 of the deaths information was available regarding medical history and medication. Ten of the deceased had been prescribed a total of 18 antipsychotic medications (1–4 per case). Toxicological analysis was performed in 52 cases. When analysis was aimed at the detection of therapeutic levels of antipsychotics, it was found six cases appeared to be compliant (antipsychotics detected were consistent with prescribed medication), one case was partially compliant (one of the four antipsychotics prescribed was detected) and three cases were considered to be non-compliant (therapeutic concentrations of antipsychotics were not detected). Post-mortem findings were not specific. No visceral petechiae or serosal haemorrhages were noted. Other findings are summarised in Table 1.

Table 1
Results of autopsy findings in heat-related and non-heat-related deaths.

	Heat-related ($n = 54$)	Non-heat-related ($n = 22$)
Sex	Male 36 Female 18	Male 13 Female 9
Age	27–93 years Mean 70.4 years	25–95 years Mean 66 years
Body Mass Index	13–62 kg/m^2 Mean 26 kg/m^2	17–39 kg/m^2 Mean 25 kg/m^2
Place of death	Indoors 42 Outdoors 10 Not recorded 2	Indoors 15 Outdoors 2 Not recorded 5
Decomposition[a]	Not present 8 Present 46	Not present 13 Present 9
Liver	Flaccid capsule 7 Severe fatty change 6 (of 47 cases) Increased fibrosis 8 (of 47 cases)	Flaccid capsule 1 Severe fatty change 3 (of 18 cases) Increased fibrosis 1 (of 18 cases)
Vitreous sodium	101–117 mmol/L (14 cases) Mean 134 mmol/L	112–157 mmol/L (10 cases) Mean 132 mmol/L
Vitreous creatinine	84–767 μmol/L (22 cases) Mean 227 μmol/L	92–578 μmol/L (7 cases) Mean 278 μmol/L
Vitreous urea	4.6–59 mmol/L (18 cases) Mean 19 mmol/L	7.1–24.9 mmol/L (7 cases) Mean 17.5 mmol/L
Vitreous glucose	0.1–10.7 mmol/L (18 cases) Mean 1.6 mmol/L	0.1–5.0 mmol/L (10 cases) Mean 1.4 mmol/L
Non-narcotic analgesic detected (eg: paracetamol, aspirin) Limit of detection 0.5 mg/L	12 cases of 52 (23%)	5 cases of 18 (22%)
Narcotic detected Limit of detection 0.01 mg/L	7 cases of 52 (13%)	2 cases of 18 (11%)
Antipsychotic Limit of detection 0.001 mg/L	7 cases of 52 (13%)	3 cases of 18 (17%)
Antidepressant Limit of detection 0.01 mg/L	7 cases of 52 (13%)	1 cases of 18 (6%)
Sedative detected (eg: diazepam) Limit of detection 0.01 mg/L	0 cases of 52	2 cases of 18 (11%)
Diuretic detected Limit of detection 0.5 mg/L	1 case of 52 (2%)	0 cases of 18
Benztropine detected Limit of detection 0.01 mg/L	2 cases of 52 (4%)	0 cases of 18
Cannabinoids detected Limit of detection 0.002 mg/L	2 cases of 52 (4%)	1 case of 18 (6%)

[a] Proportion of decomposed cases in heat-related deaths (46 of 54) significantly greater than for non-heat-related deaths (9 of 22), $p = 0.004$ chi squared test; all other comparisons not significant ($p > 0.05$).

During the period of the heat wave 32 deaths from natural causes not related to heat were reported to the Coroner, of which 22 underwent post-mortem examination. In this group the cause of death was determined as cardiac disease in 16, gastrointestinal related in 2 (one each ruptured oesophagus and acute haemorrhage), epilepsy in 2 cases, pulmonary thromboembolus in one, and metastatic melanoma in one. Of the 15 deaths that had occurred indoors (including 1 at place of work), 6 had been found dead in or on their bed. A recording of the presence or absence of air conditioning was made in 7 cases – it was present in 3 and was on in 1. The presence or absence of a fan was noted in 5 cases; in 3 a fan was present, but it was not recorded if the fan had been operating. In the 8 cases in which the interior temperature was recorded it ranged from 20 to 34 °C, or was noted as cool (2 cases), not hot (1 case) or hot (3 cases). Other findings are summarised in Table 1.

The only statistically significant difference between the two groups (heat-related deaths and non heat-related deaths) was the greater proportion of decomposed cases in heat-related deaths (46 of 54) compared to non heat-related deaths (9 of 22), $p = 0.004$ (chi squared test).

4. Discussion

A survey of the published literature indicated that findings reported in heat-related deaths are generally non-specific and non-diagnostic.[12–19] Haemorrhages may involve the brain (meninges and brain matter), kidneys, liver, heart (intramyocardial and subendocardial), lung and muscle. Coagulative necrosis may be seen in the heart. Fatty change or hepatocellular degeneration with necrosis may be found in the liver. Mucosal haemorrhages may be present in the stomach, duodenum and small intestine. The adrenal glands can be affected by cortical necrosis. There may be evidence of diffuse intravascular coagulation, with microthrombi in the kidneys. Gross haemorrhages may be seen around the renal pelves. Cerebral oedema may be present. However, petechiae and ecchymoses of serosal membranes may be the only findings in acute deaths; if there has been a period of survival rhabdomyolysis, pancreatitis, centrilobular necrosis of the liver and tubular necrosis in the kidneys may be observed. Many cases can be expected to be decomposed,[7] which will impair assessment. Careful attention should be placed toward documentation of natural diseases. Specific conditions to document include coronary artery atherosclerosis, and pulmonary disease including emphysema. In young individuals or unusual presentations, it has been recommended that the thyroid gland should be examined.[20]

Ancillary tests may assist. Biochemical analysis of the sodium level in vitreous fluid from the eyes may give an indication of dehydration.[21–23] Levels of urea may also rise due to dehydration,[24] but if urea and creatinine are elevated the possibility of renal failure[25] resulting from underlying rhabdomyolysis[26,27] or hypotension[28] should be considered and immunohistochemistry for myoglobin in kidney sections may assist with diagnosis of rhabdomyolysis.[29] Histological examination may be limited by heat-related post-mortem changes. Nonetheless, a search should be made for natural disease that may have contributed to death and for natural disease, such as pneumonia or myocarditis, which may not be apparent macroscopically.[30] Toxicological analysis may detect therapeutic levels of drugs that have the potential to contribute to heat-related death[7] including anticholinergics that may have been prescribed for urinary incontinence[31]; however, specimens may be difficult to obtain at the time of autopsy due to effects of heat including decomposition. A comparison between medications known to be prescribed to the deceased or present at the scene with the toxicological findings in this case series indicated a degree of non-compliance, which has been previously recognised.[32] Thus assessments of the possible relevance of psychiatric medication to heat-related deaths[33–35] should be based on toxicology findings, not prescribing data.

South Australia has a forensic service that is unique within Australia as the Coronial service for the entire population of the state is provided by the State Coroner's Office and Forensic Science services based in Adelaide. The state has an area of nearly 1 million square kilometres (comprising nearly 13% of the total area of Australia). The majority of the population is present in the south-eastern coastal region (predominantly within the capital: Adelaide); a large proportion of the remaining area is desert. In 2009 the State had an approximate population of 1.6 million.[36] All Coronial matters are handled by the State Coroner's Office (comprising a State and Deputy State Coroner with support staff). All Coronial directed post-mortem examinations are performed at the mortuary at the Forensic Science building in the city of Adelaide (approximately 1100–1200 autopsies per year). This has allowed a collation of all deaths possibly related to high ambient temperatures.

This review of the post-mortem findings in 54 cases of assumed heat-related deaths compared with 22 natural deaths regarded as not related to heat during the same period has confirmed that autopsy findings in heat-related deaths are generally non-specific. The observation of a wrinkled or flaccid liver capsule may be related to heat-related dehydration, but it could be a result of decomposition; this will be prospectively evaluated in the future. In this case series, the determination that a death was possibly heat-related was largely based on the reports of the circumstances, but was supplemented in some cases by biochemical evidence of dehydration.[37,38] Nonetheless, it is possible that some cases were incorrectly assigned as heat or non-heat-related.

Had testing been available, analysis for expression of heat shock proteins could have been considered in this case series as evidence of exposure to heat stress. Cellular organisms respond to heat challenge by producing proteins that have been termed heat shock proteins.[39] For example, hyperthermia will induce heat shock proteins in the kidney.[40] However, elevated levels of these proteins are not specific to heat[41] and can occur in the presence of renal disease.[42] Nonetheless, heat shock protein 70 (Hsp70) has been shown to be expressed and be protective against cell death following hyperthermia in a rat model[43] and expression of heat shock protein Hsp72 improves survival in a transgenic mouse model following hyperthermia.[44] Plasma levels of the heat shock proteins Hsp72 (HSPA1A) and Hsp27 (HSPB1) increased in response to intensity of exercise and rise in body core temperature[45] and it has been proposed that levels of Hsp-72 might be an indicator for heat stroke.[46]

Notwithstanding the possibility in future cases of biological testing for evidence of exposure to heat stress, the diagnosis of heat-related deaths is currently highly predicated on a thorough and comprehensive investigation of the circumstances and scene. Erroneous information or scene assessment can lead to incorrect diagnosis. Complicating the investigation of heat-related deaths is that the scene investigators themselves may be in harm's way from exposure to prolonged extreme temperatures and being tasked for multiple scenes of death with increased workloads. When assessing a death scene, investigators should give regard to the following factors.

- *Air conditioning.* Multiple studies[47,48,32,49] have demonstrated that air conditioners are the most important feature associated with survival during periods of hot weather. Additionally, having access to an air-conditioned space such as a lobby in a public place is also associated with increased survival.[50] A meta-analysis evaluating many heat waves showed that having

working air-conditioning or access to other air conditioned environments was associated with lower risk of death.[51]

- *Electric Fans.* Fans have been the subject of debate in the heat-related death literature with no clear evidence to support or refute a beneficial effect during heat wave conditions.[52] Some studies did not detect any reduction in mortality from the use of electric fans[50,32]; whereas, a meta-analysis demonstrated a trend toward lower risk of death that was not statistically significant.[51]
- *Baths/other cooling.* Taking baths or cool showers was shown in a meta-analysis to trend towards lower risk of death but was not statistically significant.[51] Water restrictions may lead to reluctance to use water during heat waves.
- *Situation.* Living at the top floor of a building or an upstairs room was also shown to have increased risk of death in the Chicago and Philadelphia heat waves.[53,50]
- *Mental illness.* Mental illnesses has been shown to increase risk of death during a heat wave.[50,54,32,49] In this group, heat-related death was associated with having been prescribed psychotropic and anticholinergic drugs.[32] However, newer psychiatric medications may have less toxic properties and interfere less with heat dissipation.[54] Those with mental illness may have poor situational awareness and may not watch the news, or have decreased social contacts to make them aware of an impending heat wave; associated socio-economic factors may have the consequence of less access to air conditioning.[32] The behavioural response to heat (such as wearing light clothing) may not be appropriate.[55] Cognitive impairment was found to be significantly associated with heat stroke in an Australian study.[56]
- *Medical conditions (including being bedridden).* The National Association of Medical Examiners (NAME) Ad Hoc committee on the definition of heat-related fatalities notes that a significant number of deaths occur in persons known to have a pre-existing disease that is worsened by heat stress.[7] A meta-analysis disclosed an increased risk of death with cardiovascular and pulmonary illness.[51] Other predisposing conditions include cerebrovascular disorders.[6] Fatal heat stroke has been described in a young woman with previously undiagnosed Hashimoto's thyroiditis, and the thyroid should be considered in unusual cases of heat stroke.[20] Those who are confined to bed were shown to have the worst negative predictive factor for death in a heat wave study from Chicago.[50] Being bedridden may interfere with heat dissipation, and the individual may not activate air conditioning or be able to enter an air-conditioned space. These individuals may have medical conditions that can predispose to death during heat waves.
- *Subject constitution.* Extremes of age, including the young and elderly may be particularly susceptible to heat. Obese individuals are susceptible, but those with a body-mass-index (BMI) below the normal range may also have an increased risk of death.[50]
- *Social isolation.* Limited social contact is a risk factor, because the individual may not be aware of the impending seriousness of the situation and may be unable to seek help. However, meta-analysis evaluation demonstrated that although social isolation was associated with a trend toward increased risk of death, it was not a statistically significant factor.[51]
- *Activity around the time of death.* Sustained, strenuous exertion is a recognised cause for hyperthermia even in fit healthy individuals.[57–59]
- *Alcohol.* It is advised to avoid alcohol during a heat wave, with general agreement that it is a predisposing factor to heat stroke.[60] Additionally, individuals with chronic alcohol abuse may have issues with self-care and decreased situational awareness regarding an impending heat wave, similar to those with mental illness. Acute alcohol intoxication and alcoholic liver disease have been reported to be associated with deaths caused by exposure to high environmental temperatures within Australia.[26]
- *Medications.* Numerous medications may have side effects, which may impair survival in extreme heat conditions, classically these are the antipsychotic medications with anticholinergic side effects.[54,61] Anticholinergics can decrease sweating and therefore heat dissipation.[62] In addition, medications such as phenothiazines, can affect central thermoregulation.[62] Mepazine, known to be the most anticholinergic of all antipsychotic medications was withdrawn from use because of reports of heat stroke.[63] Clozapine and olanzapine have known anticholinergic properties, and clozapine is known to increase body temperature.[54] Heat intolerance may be associated with certain antidepressant medication.[64] Careful attention should be placed to medical conditions and medications, which ordinarily would not concern the forensic pathologist. For example, urinary retention would ordinarily not be of concern in the pre-mortem evaluation prior to an autopsy. However, anticholinerigics such as oxybutynin used in the treatment of urinary retention can interfere with heat loss through sweating, resulting in a risk for heat-related illness.[65,31] Multiple other medications can also predispose to heat stroke or heat-related death,[66] including diuretics with a particularly high risk in the elderly.[67,56] However, results of toxicological analysis of victims of heat indicated that non-compliance with prescribed medication was common thus observations of medication at the scene may not be relevant to the cause of death.[32]
- *Power interruptions.* Interruption of electricity is not uncommon during heat waves. The National Association of Medical Examiners (NAME) position paper on heat-related deaths describes one case in which the electricity had been out for 5 h because of a power failure. The cause of death in that case was arteriosclerotic cardiovascular disease with heat stress as a significant contributing factor.[7] A power failure may have contributed to the death of an elderly man during a period of high ambient temperature ($>32\ ^{\circ}C$).[68]
- *Factors unique to the Australian environment.* In a report of deaths of four tourists in South Australia, lack of familiarity with the harsh Australian conditions was considered to have been a contributory factor.[26]

This emphasises that a thorough scene assessment is an essential component of the investigation of possible heat-related deaths. However, the assessment was sub-optimal in a number of cases. For example, it was unknown what activities deceased had been engaged in around the time of their death such that any contribution from exertion induced hyperthermia could not be assessed. In the future, a pre-formulated work sheet with appropriate questions to be answered at the time of the scene investigation would assist investigating officers and forensic pathologist. A suggested guideline is shown in Table 2. This approach can prevent ambiguous or misleading information, such as an air conditioner being present at the scene without information regarding if it was functioning. The importance of measurement of temperature at the scene with avoidance of subjective descriptors should be stressed.

In conclusion, a full post-mortem examination with use of ancillary tests is unlikely to result in findings that would conclusively diagnose a heat-related death, but could reveal a natural or other cause of death not related to heat exposure. As heat-related deaths may be defined as "a death in which exposure to high ambient temperature either caused the death or significantly

Table 2
Sample scene investigator work sheet for possible heat-related deaths.

Case reference:
Date body found:
Date last seen:
Circumstantial evidence regarding possible date of death (eg: newspapers):
Location/setting found (eg: indoors, outside):
Where body found (eg: in bed, on floor):
Body position (eg: face up):
Body coverings/clothing (eg: sheets or bedding):
Air conditioner Yes/No
Air conditioner switched on or off ? Air conditioner working/effective? (irrespective of whether on or off)
Evidence of other cooling methods (eg: fan, water baths):
Environmental temperatures for period of suspected death (consult meteorological data):
House temperature:
Evidence of type of activity prior to death (eg: resting/gardening):
Medications present?
Evidence of drug or alcohol use?
Known medical conditions (including psychiatric illness):
Lives alone?
Was power interrupted ? If yes, date and duration:
If treated at hospital prior to death, was there an ante-mortem temperature:

contributed to it",[7] a reproducible method of indicating an abnormally high temperature is required. In this regard, calculation of the Excess Heat Factor is proposed as a method for identifying heat waves that may be associated with excess mortality.[11] Although calculation of the Excess Heat Factor does require assistance from a meteorologist it does allow for identification of severe heat wave events, which can be defined by Excess Heat Factor values that exceed thresholds for the region and extreme heat wave events when the Excess Heat Factor is much higher than the severity threshold.[1] A good scene examination is required, which may be assisted by providing investigators with a work sheet indicating what information is required.

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